**U.S. Department of Labor**

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**Issue Date: 01 May 2006**

CASE No. 2005-BLA-5331

In the Matter of

IRMA MARIE HALLER,
SURVIVOR OF ALPHONSE R. HALLER,
Claimant

v.

CANTERBURY COAL COMPANY,
Employer

and

INTERNATIONAL BUSINESS &
MERCANTILE REASSURANCE COMPANY,
Carrier

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,
Party-in-Interest.

Appearances:

Anthony Kovach, Esquire
For the Claimant

George H. Thompson, Esquire
For the Employer

Before: MICHAEL P. LESNIAK
Administrative Law Judge

## DECISION AND ORDER – AWARDING BENEFITS

This proceeding arises from a widow's claim for survivor's benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 *et seq.* (the Act). The Act and implementing regulations,

20 C.F.R. Parts 410, 718, and 727 (Regulations), provide compensation and other benefits to coal miners who are totally disabled by pneumoconiosis and to the surviving dependents of coal miners whose death was due to pneumoconiosis.

The Act and Regulations define pneumoconiosis (commonly known as black lung disease, coal workers' pneumoconiosis, or CWP) as a chronic dust disease of the lungs and its sequelae, including respiratory and pulmonary impairments arising out of coal mine employment. 20 C.F.R. § 725.101.

## PROCEDURAL HISTORY

The miner filed four claims for benefits with the Department of Labor (DOL). His first claim, filed on August 9, 1982, was denied by Administrative Law Judge George Morin on April 20, 1988. Judge Morin found the miner worked almost 34 years in coal mine employment. Because the pulmonary function study and blood gas evidence were non-qualifying, Judge Morin credited the physicians' reports which concluded the miner did not have pneumoconiosis and was not totally disabled due to pneumoconiosis. The miner filed again on February 22, 1993, and the OWCP denied that claim on August 18, 1993. The miner did not pursue that denial. He filed a third claim for benefits on August 28, 1996, which OWCP denied on December 31, 1996, and the miner did not pursue this denial. On September 1, 1999, the miner filed his fourth claim for benefits. Administrative Law Judge Daniel Leland issued a denial on February 7, 2002. Judge Leland also found the miner worked almost 34 years in coal mine employment. Judge Leland found the chest x-ray evidence was positive for pneumoconiosis, however, he found the persuasive medical opinion reports were negative for pneumoconiosis. On consideration of all of the evidence, Judge Leland found claimant-miner had not established the presence of pneumoconiosis. Judge Leland also found the pulmonary function study and blood gas study results were non-qualifying and there was no evidence of cor pulmonale, so he also found the claimant-miner had not established total disability. Accordingly, benefits were denied on February 7, 2002.

The miner died on November 8, 2003, and his widow filed a claim for survivor's benefits on December 16, 2003. (DX 2)[1]. The District Director issued a Proposed Decision and Order on September 16, 2004 in which he awarded the claim because the evidence, specifically the autopsy report, established the presence of pneumoconiosis and that the miner's death was due to pneumoconiosis. (DX 27). Employer objected to the findings of the District Director and requested a formal hearing before an ALJ on September 21, 2004 and again on October 21, 2004. (DX 27, 28).

On November 1, 2005, I held a hearing in Pittsburgh, Pennsylvania. The Claimant and Employer, both represented by counsel, were afforded the full opportunity to present evidence and argument. I admitted Director's Exhibits 1–10 and 12-35, Claimant's Exhibits 1-10, and Employer's Exhibits 1 and 2. (TR 7-9). Director's Exhibit 11 was excluded as it was duplicative of the documents included in Director's Exhibit 15.

[1] The following abbreviations are used in this opinion: DX = Director's exhibit, EX = Employer's/Carrier's exhibit, CX = Claimant's exhibit, TR = Transcript of the November 1, 2005 hearing.

The parties stipulated to Employer's proper designation as the Responsible Operator and to at least 34 years of qualifying coal mine employment by the deceased miner. In addition, the parties stipulated that the Claimant is the surviving spouse. (TR 9-10).

## ISSUES

1. Whether the miner had pneumoconiosis;

2. Whether the miner's pneumoconiosis arose out of his coal mine employment; and,

3. Whether the miner's death was due to pneumoconiosis as provided by § 718.205 (c).

## FINDINGS OF FACT

Length of Coal Mine Employment

The parties agree, and I find that the evidence of record establishes that the miner was a coal miner within the meaning of the Act and Regulations for at least thirty-four years. (TR 10, DX 4, 5, 6, 8).

Claimant's Testimony

The Claimant testified at the hearing that she has not remarried since her husband's death. (TR 12). The record includes a marriage certificate which establishes Claimant, Irma Marie Wilkinson, married the deceased miner, Alphonse R. Haller, on October 25, 1941. (DX 9).

Medical Evidence

The record includes the evidence submitted with the miner's four previous claims for benefits. However, pursuant to the Board's holding in *Church v. Kentland-Elkhorn Coal Corp.*, BRB Nos. 04-0617 BLA and 04-0617 BLA-A (Apr. 8, 2005) (unpub.), medical evidence submitted in a living miner's claim is not automatically admissible in a survivor's claim filed after January 19, 2001. Rather, in a survivor's claim, the medical evidence from the prior living miner's claim must be designated as evidence by one of the parties in order for it to be included in the record relevant to the survivor's claim.

The following evidence has been designated as evidence by one of the parties:

Chest X-ray

| Exh.# | X-ray Date | Physician/Qualifications | Interpretation |
|---|---|---|---|
| CX 9 | 10-01-99 | Lynn, BCR, B | 1/0 s, t |
| CX 10 | 10-01-99 | Gaziano, BCR, B | 1/0 s, t |

Pulmonary Function Studies

No new pulmonary function studies were submitted.

Arterial Blood Gas Studies

Any new blood gas studies included in the hospital records that were submitted with the survivor's claim were taken when the miner was in the hospital and while he was receiving supplemental oxygen.

Physicians' Reports

Records from Dr. S. Bajwa, the miner's treating physician, establish the miner was diagnosed with lymphoma in June, 2002. In 2001 and 2002, Dr. Bajwa's notes included symptoms of shortness of breath. Pneumoconiosis and chronic obstructive pulmonary disease were listed as diagnoses in reports prepared between 2000 through 2003. Dr. Bajwa also treated the miner for uncontrolled diabetes and for controlled diabetes over this same time period. (DX 13, 14).

The miner was hospitalized at the Allegheny Regional Hospital in May, 2002 when dissection of his left neck revealed probable lymphoma. He was hospitalized again in February, 2003 for uncontrolled diabetes. Reports from February, 2003 note the miner was being treated with chemotherapy for lymphoma. The miner was hospitalized from June 26 to July 2, 2003 for treatment of dehydration and control of his blood sugar. The miner was again transferred from the nursing home to the hospital from September 3 to 6, 2003, for treatment of poor eating, vomiting, nausea, and diarrhea. Then from September 15 to 18, 2003, Dr. Bajwa treated the miner at the Allegheny Regional Hospital for anemia. On all these hospitalizations, the diagnosis list included pneumoconiosis and chronic obstructive pulmonary disease. (DX 14).

The miner was hospitalized at Forbes Regional Hospital from September 28 to October 5, 2003 and treated for acute respiratory failure. The admission list included pneumonitis due to inhalation of food or vomitus, staphylococcal aureus septicemia, decubitus ulcer, unspecified protein-calorie malnutrition, diabetes mellitus without complication, type 1, chronic obstructive pulmonary disease, infection which was resistant to penicillin, hypotension, essential hypertension, anemia, depressive disorder, and esophageal reflux. A feeding tube was inserted because the miner had dementia and was unable to eat. He was discharged to Select Care.

(EX 1). Reports from Select Specialist Hospital indicate the miner was admitted on October 5, 2003 with aspiration pneumonia and was treated with bronchodilators, supplemental oxygen, IVs, and diabetic meals. A consultation on November 8, 2003 stated the miner had pneumonia, respiratory failure, chronic obstructive pulmonary disease, c-difficile colitis, diabetes mellitus, anemia, and depression. Dr. Wodzinski stated at this time, the patient's prognosis is poor and survival beyond that day was doubtful. (DX 12).

The miner died on November 8, 2003. The miner's death certificate, signed by Dr. D. Robinson-Menzie, listed the immediate cause of death as respiratory failure, due to chronic obstructive pulmonary disease, pneumonia MRSA, and pneumoconiosis. The death certificate noted C-difficle colitis as another significant condition. (DX 10).

Dr. C. Wecht, a board certified pathologist, performed an autopsy on November 9, 2003. Dr. Wecht reported numerous emphysematous blebs and bullae in the upper lobes of the lung and nodules on the pleural surface with black anthracotic streakings noted profusely over the surface of both lungs. Cut sections of the lung showed the same pattern of small nodules and black carbonaceous pigment but to a lesser degree than on the surface. On microscopic examination, the findings were consistent with the gross observations. Dr. Wecht's final pathological diagnosis was:

Arteriosclerotic Cardiovascular Disease:
- Atherosclerosis of coronary arteries, aorta and cerebral arties, severe.
- Myocardial infarction, old, healed, left ventricle.
- Myocardial fibrosis, focal, microscopic.

Chronic Obstructive Pulmonary Disease:
- Anthracosilicosis (Coal Workers' Pneumoconiosis)
- Pulmonary emphysema, bilateral
- Pulmonary fibrosis, bilateral.
- Fibroanthracotic and fibrohyaline macules and micronodules.
- Fibroanthracosis and fibrohyalinization of mediastinal and peribronchial lymph nodes.
- Cor pulmonale.
- Increased anteroposterior diameter of the chest.
- Pulmonary osteoarthropathy.

Lymphoma evidence in periaortic and mediastinal lymph nodes, small cell type
Acute bronchopneumonia, focal
Chronic passive congestion of spleen
PEG insertion site
Chronic passive congestion of liver, mild
Arteriolar nephrosclerosis.
Chronic pyelonephritis
Chronic hemorrhagic cystitis, focal
Status, post-prostatectomy.
Dehydration
Malnutrition

On June 9, 2004, Dr. Wecht reviewed the autopsy and medical records. He stated the miner's pneumoconiosis was present prior to his death and the pulmonary pathology at death was coal workers' pneumoconiosis. He stated this finding was based on the clinical, medical, and social history as confirmed by autopsy. Dr. Wecht stated the miner's death was due to arteriosclerotic cardiovascular disease. He stated further that coal workers' pneumoconiosis, which was the basis for the chronic obstructive pulmonary disease, was a substantially contributing factor in the miner's death. Dr. Wecht explained that the microscopic slides showed diffuse depositions of black anthracotic pigment in periobronchiolar and periarteriolar lymphatics. In addition, ruptured alveolar walls, consistent with extensive pulmonary emphysema, focal pulmonary fibrosis, thickening of the pleura with subplerual deposits of anthracotic pigment, anthracotic and fibrohyaline macules and micronodules, and fibroanthracosis and fibrohyalinization of mediastinal and lymph nodes were also noted. Dr. Wecht also stated examination of the lung tissue under polarized light revealed scattered birefringent crystals, consistent with silica. All these findings in the lung are compatible with the clinical diagnosis and ante-mortem history of pneumoconiosis. (DX 15, CX 1).

Dr. Wecht testified extensively at a deposition taken on June 9, 2004. He stated the presence of hypoxia in a person, such as the miner, with arteriosclerotic cardiovascular disease was dangerous because the hypoxia was putting additional stress on already diminished oxygenation due to compromised coronary vessels, atherosclerotic plaques, and calcification debris. Thus, with physical activity which required more oxygen or the presence of a disease which limited the oxygen, the miner's already stressed heart was much more susceptible.

Dr. Wecht stated the nodules and densities of coal workers' pneumoconiosis were actually felt on gross examination of the lung. He explained the presence of the nodules shows that the carbonaceous pigment and silicacious compounds had actually produced changes in the miner's lung function which were seen as microscopic scarring. Thus, he stated, the disease of coal workers' pneumoconiosis is present. The autopsy findings correlated with the clinical findings as listed in the hospital records of coal workers' pneumoconiosis. Dr. Wecht discussed the miner's other medical problems including the renal problems, diabetes, and recently diagnosed lymphoma. He noted the hospital records sometimes included the diagnosis of pneumoconiosis and sometimes the diagnosis of chronic obstructive pulmonary disease. In addition, he noted the miner had infectious pneumonia from time to time. Dr. Wecht noted that the miner smoked 10 to 15 years but that he quit in 1963. He also noted 34 years of coal mine employment.

Dr. Wecht stated there was no evidence that the miner had any pulmonary emphysema at the time he quit smoking. Although the miner could have had some minimal emphysema due to his smoking history, it was Dr. Wecht's opinion the emphysema at autopsy, if not completely then overwhelmingly, was due to coal mine dust exposure. Dr. Wecht opined that the emphysema was a component of pneumoconiosis.

Dr. Wecht stated the principle diagnoses at autopsy were the arteriosclerotic heart disease with severe atherosclerosis of the coronary arteries, aorta and cerebral arteries, and the old myocardial infarction. Dr. Wecht noted lymphoma was present at autopsy but that it did not play a significant role in the miner's death. He stated the miner developed pneumonia terminally and

had kidney changes, was dehydrated, and was malnourished. Dr. Wecht stated the coal workers' pneumoconiosis present was a secondary disease process which was not directly related to the arteriosclerotic heart disease. He agreed that coal mine dust did not cause the arteriosclerotic heart disease. He stated it was difficult to grade the coal workers' pneumoconiosis, but he estimated that on a scale of 1 to 10, the coal workers' pneumoconiosis present was at 7.

Dr. Wecht explained, however, the change in the lungs made the heart work harder, and he discussed the findings of cor pulmonale, including the disproportionately enlarged right heart. In addition, he noted the autopsy found increase in the anterior posterior diameter of the chest and a widening or thickening of the phalanges which was listed as pulmonary osteoarthropathy. These two findings also established the presence of a serious pulmonary disease. Dr. Wecht stated the miner had two disease processes with two organ systems that aggravated one another. He agreed with the death certificate that the immediate cause of death was respiratory failure due to pneumonia, chronic obstructive pulmonary disease, and pneumoconiosis. He stated pneumoconiosis contributed to the miner's death because the miner's bad heart was worsened by the pneumoconiosis. In addition, he noted the miner's situation with pneumoconiosis and heart problems made him more likely to develop an infection, such as pneumonia which led to respiratory failure and ultimately cardiac failure.

On cross examination, Dr. Wecht stated again the primary cause of death was the arteriosclerotic heart disease with severe coronary atherosclerosis. He stated again this cardiac problem was not caused by exposure to coal mine dust. Dr. Wecht agreed that a person could get shortness of breath and hypoxemia from coronary artery disease; however, he noted that would occur with both left and right-sided heart enlargement. Because in this case, the miner's right ventricle was enlarged disproportionately and the miner's heart was not enlarged overall, Dr. Wecht stated this established the miner did not have hypertensive heart disease. Dr. Wecht agreed that chronic passive congestion in the lungs, spleen and liver were present. However, Dr. Wecht opined that chronic congestive heart failure was not a significant etiologic factor in the right sided heart enlargement. Dr. Wecht also agreed on cross examination that progressive massive fibrosis was not present nor was complicated pneumoconiosis. He stated he was not surprised that the miner's coal workers' pneumoconiosis was not seen on chest x-ray because the nodules were only 2 to 3 millimeters in size and the chest x-ray would not see nodules unless they were 5 millimeters in size. Dr. Wecht stated that because the emphysema present was in the upper lobes, it would not be called panlobular emphysema. He agreed that cigarette smoking is a cause of emphysema, but in a person without any smoking history for 40 years, a remote smoking history does not explain the emphysema present. Dr. Wecht did not report any findings of asthma on autopsy; however, he stated that is fairly standard because asthma is primarily a clinical diagnosis.

He stated it was likely the miner had cardiac arrhythmia which led to cardiac arrest and then to cardiopulmonary failure. However, he also stated cardiac arrhythmia does not produce evidence which can be seen at autopsy. While Dr. Wecht stated he could not estimate how much longer the miner would have lived without the presence of the long standing lung disease which produced a continuous drain and increased the burden on the heart, he stated the miner would have lived a few or several months or perhaps even a few years. Dr. Wecht reiterated this lung disease added an extra burden on the miner's already badly compromised heart and slowly and

insidiously compromised his overall condition. Dr. Wecht agreed the diabetes present also made the miner more susceptible to infection and led to renal failure. He also stated that there was no evidence of aspiration pneumonia on autopsy. (CX 3).

Dr. E. Oesterling, a board certified pathologist, reviewed the evidence including the histologic slides of which he made some photomicrographs on July 20, 2004. Dr. Oesterling stated the slides showed a level of change in the lungs sufficient to diagnose mild to moderate macular with focal pleural micronodular coal workers' pneumoconiosis. Dr. Oesterling stated the low level of the disease meant it would have had little impact on the miner's pulmonary function and, without greater structural change, a disability during the miner's lifetime due to pneumoconiosis is unlikely. Dr. Oesterling stated without more significant tissue alteration, the coal workers' pneumoconiosis present would have not impacted upon or caused the miner's death.

Dr. Oesterling stated the slides show that another significant respiratory disease was also present, which accounts for the miner's respiratory disability during his life, and he stated that the respiratory disease was moderately severe panlobular pulmonary emphysema. Dr. Oesterling stated this respiratory disease was not due to coal mine dust exposure because coal mine dust was not associated with the sections of emphysema. Rather, he stated it was due to the miner's history of asthma, previous smoking history, and age. Dr. Oesterling stated the report at autopsy of the development of bullous emphysema demonstrated a further progression of the panlobular emphysema. Dr. Oesterling stated the slides demonstrated the miner had also developed pulmonary hypertension and vasculitis as well as cor pulmonale. These conditions were due to the panlobular emphysema and were not due to coal mine dust exposure. In addition, he noted the presence of acute bronchopneumonia which caused additional obstruction of the airways and further destruction of the lungs. This diagnosis was a critical factor in the miner's final days. Dr. Oesterling stated the interstitial fibrosis with multiple foci represented dystrophic calcification and ossification, a common finding in scarred lungs, and was also unrelated to coal mine dust exposure.

Dr. Oesterling stated there was clear evidence of left-sided heart failure in the medical record and in the lung slides. He noted the disease impacted other organs and systems, including the kidneys. Dr. Oesterling also stated the kidneys were impacted by the miner's poorly controlled hypertension and poorly controlled diabetes. In addition, he noted changes in the spleen and lymphoma nodes, specifically the small cell lymphatic lymphoma, which also depleted the miner's immune system and made him predisposed to the development of pneumonia.

Dr. Oesterling concluded: 1) the miner had significant underlying chronic obstructive pulmonary disease, moderate to severe, panlobular progressing to bullous pulmonary emphysema; 2) the miner's chronic obstructive pulmonary disease was due to history of asthma, remote smoking history, and aging; 3) the miner's chronic respiratory status was further compromised by the development of bronchopneumonia; 4) the miner's interstitial scarring within the lower lobes was due to emphysema and chronic passive congestion resulting in dystrophic calcification and ossification; 5) the miner had significant coronary and generalized arteriosclerosis; 6) the arteriosclerotic changes resulted in significant remote and progressive

ischemia, and destruction of the left ventricular wall; 7) arteriosclerosis and diabetes resulted in significant destruction of the kidneys through nephrosclerosis and chronic pyelonephritis; 8) acute renal failure resulted which impacted the miner's cardiac status; 9) the miner also had small cell lymphocytic lymphoma with resultant chronic lymphocytic leukemia; 10) none of the aforementioned diagnoses were due to coal mine dust exposure; 11) the miner had changes due to coal mine dust exposure which results in mild to moderate macular with focal pleural micronodular coal workers' pneumoconiosis; 12) the level of coal workers' pneumoconiosis present did not cause respiratory disability; and, 13) the limited coal workers' pneumoconiosis present was insufficient to have contributed to or hastened or caused the miner's death. (DX 16).

At a deposition taken on August 2, 2005, Dr. Oesterling stated the lung slides prepared by Dr. Wecht were an excellent and generous sampling of the miner's lungs. Dr. Oesterling stated the miner's death was a cardio-respiratory death. He noted the miner had very significant changes in his heart and very significant changes in his lungs, but he reiterated his opinion that none of the significant changes was due to coal mine dust exposure. Dr. Oesterling stated again that the miner did have coal workers' pneumoconiosis but that the interstitial changes from coal workers' pneumoconiosis were insufficient to have altered the structure of the lungs so there was no functional alteration and no symptomatology from coal workers' pneumoconiosis and it was not a factor in the miner's death.

Dr. Oesterling reiterated his written opinion that the significant chronic pulmonary disease the miner had was panlobular emphysema which was quite severe. Dr. Oesterling discussed at some length the miner's extensive medical problems, including the severe cardiac disease, ossification the base of the lungs, renal vascular disease, and diabetes. He stated the cardiovascular disease was due to arteriosclerosis and the diabetes present accelerated the disease. As the disease progressed, the heart muscle fibers were more and more destroyed, and the miner progressed into congestive heart failure. The panlobular emphysema was not due to coal mine dust exposure because dust was not present in the areas of emphysema. In addition, the development of panlobular progressing into bullous emphysema is not typically seen with mild macular focal pleura nodular coal workers' pneumoconiosis. Rather, these kind of changes are seen with progressive massive fibrosis or complicated pneumoconiosis. Dr. Oesterling also quoted a British book which links centriacinar emphysema to coal mine dust exposure but not panlobular emphysema. Dr. Oesterling stated there are two main causes for emphysema, asthma and smoking, and the miner had both of these in his history. In addition, Dr. Oesterling stated older people sometimes develop senile emphysema which is related to a loss of elasticity in the lungs as a person ages.

Dr. Oesterling also discussed the fact that the nodular coal workers' pneumoconiosis was present in the interstitium, but to a lesser degree than in the pleura. The interstitium is where the lung function actually takes place, so the lesser degree of coal workers' pneumoconiosis present in this part of the lung was significant. The emphysema, passive congestion, developing pneumonia, and lymphoma all reduced the capacity in the lungs which were already scarred by panlobular emphysema. Thus, the miner became hypoxic which led to his decline.

Dr. Oesterling also stated it was his opinion the miner's right ventricle was not markedly enlarged, so he did not think those changes were significant. He did agree that the miner had

pulmonary hypertension and vascular changes, which are the precursors for cor pulmonale. Dr. Oesterling stated again, it was his opinion these changes were due to emphysema and not to coal mine dust exposure. Dr. Oesterling agreed the miner's emphysema was a factor in his death, especially with the development of the pneumonia and with the presence of passive congestion. Dr. Oesterling also agreed the miner had no active asthma at the time of his death. (EX 2).

## CONCLUSIONS OF LAW

### Entitlement to Benefits

This claim must be adjudicated under the regulations at 20 C.F.R. Part 718 because it was filed after March 31, 1980. The regulations provide a survivor claimant must establish, by a preponderance of the evidence, that the miner had pneumoconiosis, that his pneumoconiosis arose from coal mine employment, and that his death was due to pneumoconiosis. 20 C.F.R. § 718.205. The Part 718 regulations provide that a survivor is entitled to benefits only where the miner's death was due to pneumoconiosis. 20 C.F.R. § 718.205(a). As a result, the survivor of a miner who was totally disabled due to pneumoconiosis at the time of death but died due to an unrelated cause is not entitled to benefits. Under § 718.205(c)(4), if the principal cause of death is a traumatic injury or a medical condition unrelated to pneumoconiosis, the survivor is not entitled to benefits unless the evidence establishes that pneumoconiosis was a substantially contributing cause of the death. The regulations also provide that in a Part 718 survivor's claim, the Judge must make a threshold determination as to the existence of pneumoconiosis arising out of coal mine employment, under 20 C.F.R. § 718.202(a), prior to considering whether the miner's death was due to the disease under § 718.205. 20 C.F.R. § 718.205(a).

### Existence of Pneumoconiosis

30 U.S.C. § 902(b) and 20 C.F.R. § 718.201 define pneumoconiosis as "a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment." The definition is not confined to "coal workers' pneumoconiosis," but also includes other diseases arising out of coal mine employment, such as anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, progressive massive fibrosis, silicosis, or silicotuberculosis.[2] 20 C.F.R. § 718.201. The term "arising out of coal mine employment" is defined as including "any chronic pulmonary disease resulting in respiratory or pulmonary impairment significantly related to or substantially aggravated by dust exposure in coal mine employment."

Pursuant to § 718.202, a living miner can demonstrate the presence of pneumoconiosis by: 1) x-rays interpreted as being positive for the disease; 2) biopsy evidence; 3) the presumptions described in §§ 718.304, 718.305, or 718.306, if found to be applicable; or

[2] The regulations define "pneumoconiosis" as a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or "clinical", pneumoconiosis and statutory, or "legal", pneumoconiosis.

4) a reasoned medical opinion which concluded the disease is present, if the opinion is based on objective medical evidence such as blood-gas studies, pulmonary function studies, physical examinations, and medical and work histories.

In its brief, the Employer conceded the presence of pneumoconiosis. This stipulation is well supported by the positive chest x-ray reports submitted with the widow's claim for benefits, the findings of coal workers' pneumoconiosis on autopsy by Dr. Wecht, the hospital and medical treatment records which include the diagnosis of coal workers' pneumoconiosis, and the review report by Dr. Oesterling which also concluded coal workers' pneumoconiosis was present. There is no evidence which contradicts these findings. Thus, the presence of "medical" coal workers' pneumoconiosis is clearly established under § 718.202.

Dr. Wecht and Dr. Oesterling disagreed as to the etiology of miner's emphysema. Dr. Wecht opined that miner's emphysema was due to coal mine dust exposure and pneumoconiosis. Dr. Oesterling opined that emphysema was due to asthma, smoking, and aging. The two reports were prepared by equally qualified physicians. Both physicians were thorough and detailed in discussing the findings on autopsy, the medical record, and the basis for their conclusions regarding the changes in the miner's lungs. After considering these opinions carefully, I find Dr. Wecht's opinions more persuasive. Dr. Wecht's finding that the emphysema was due to coal mine dust exposure and pneumoconiosis was based in part on the fact that the miner had stopped smoking over 40 years ago and that there was no pulmonary emphysema present at that time. While Dr. Wecht noted that some part of the emphysema may be due to the miner's remote smoking history, his opinion that the miner's more recent coal mine dust exposure was responsible for an overwhelming part of the emphysema. Thus, Dr. Wecht concluded, miner's emphysema was a component of the coal workers' pneumoconiosis diagnosed by the nodular changes as is well supported by his findings on autopsy. In addition, Dr. Wecht stated the emphysema present would not be panlobular emphysema because it was present in the miner's upper lungs.

Moreover, I note that Dr. Oesterling based his opinion that the emphysema was not due to coal mine dust exposure, in part, on this finding the miner had a history of asthma. Although one medical report mentioned asthma, the vast majority of the medical reports listed chronic obstructive pulmonary disease and pneumoconiosis as the diagnoses, including many of the treatment reports from Dr. S. Bajwa. Thus, I find Dr. Oesterling's opinion based on a finding of asthma is not well supported by the record.

I have considered the fact that Dr. Bajwa was the miner's treating physician. His opinion must, therefore, be considered pursuant to § 718.104(d). This regulation states that the relationship between the treating physician and the miner may constitute "substantial evidence" towards assigning controlling weight to a treating physician's opinion, provided it is credible "in light of its reasoning and documentation, other relevant evidence and the record as a whole." § 718.104(d)(5). The regulations list several factors to be considered including the nature of the relationship, the duration of the relationship, the frequency of the treatment, and the extent of the treatment. 20 C.F.R. § 718.104(d). In this case, the treatment records and hospital records establish Dr. Bajwa treated the miner during frequently and extensively from at least 2000 through his death in 2003. As noted above, Dr. Bajwa included a diagnosis of chronic

obstructive pulmonary disease/pneumoconiosis on most of the treatment and hospital records. Because Dr. Bajwa's conclusions are well supported by the autopsy findings of Dr. Wecht, I find Dr. Bajwa's report to be entitled to great weight. Furthermore, I find the combined weight of the autopsy report by Dr. Wecht and the findings by Dr. Bajwa, the miner's treating physician, outweigh the contrary findings by Dr. Oesterling. Therefore, I find the persuasive evidence establishes the miner's pulmonary emphysema was also due to coal mine dust exposure. Thus, I find these persuasive opinions establish the presence of "legal" pneumoconiosis under the provisions of § 718.204(a)(4).

Cause of Pneumoconiosis

Once the miner is found to have pneumoconiosis, the claimant must show that it arose, at least in part, out of coal mine employment. 20 C.F.R. § 718.203(a). If a miner who suffered from pneumoconiosis was employed for ten years or more in the coal mines, there is a rebuttable presumption that the pneumoconiosis arose out of such employment. 20 C.F.R. § 718.203(b). Because the miner had 34 years of coal mine employment, Claimant receives the rebuttable presumption that his pneumoconiosis arose out of coal mine employment. There is no contradictory evidence regarding the etiology of the "medical" pneumoconiosis. For the reasons set forth above, I find the medical opinions of Drs. Wecht and Bajwa regarding the etiology of the miner's "legal" pneumoconiosis outweigh the contrary findings of Dr. Oesterling. Therefore, I find the presumption that the miner's "legal" pneumoconiosis was also due to coal mine dust exposure is not rebutted. I find Claimant has established the miner's pneumoconiosis, both medical and legal, arose from his coal mine employment.

Death due to Pneumoconiosis

Subsection 718.205(c) applies to survivor's claims filed on or after January 1, 1982 and provides that death will be due to pneumoconiosis if any of the following criteria are met:

> (1) competent medical evidence established that the miner's death was caused by pneumoconiosis; or
>
> (2) pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or the death was caused by complications of pneumoconiosis; or
>
> (3) the presumption of § 718.304 [complicated pneumoconiosis] is applicable.

The amended regulations provide that pneumoconiosis is a 'substantially contributing cause' of a miner's death if it hastens the miner's death. 20 C.F.R. § 718.205(c)(5).

There is no evidence of complicated pneumoconiosis and, therefore, § 718.205(c)(3) is not applicable to this case. Similarly, no evidence establishes the miner's death was caused by pneumoconiosis and, therefore, § 718.205(c)(1) is not applicable to this case.

All the physicians agreed the miner's pulmonary emphysema contributed to his death. While Dr. Oesterling disagreed as to the etiology of the changes he attributed to the pulmonary emphysema, he agreed that pulmonary emphysema did contribute to the miner's death. Because I find that the more persuasive reports of Drs. Wecht and Bajwa establish the pulmonary emphysema was due to the miner's coal mine dust exposure and because all physicians agreed it contributed to the miner's death or hastened his death, I find the medical evidence does establish the miner's death was due to pneumoconiosis under the provisions of § 718.205(c)(2). In that regard, Dr. Wecht's thorough and detailed explanation of the relationship of the miner's pulmonary process to his cardiac problems as well as the relationship of the pneumoconiosis to the development of the terminal case of pneumonia and to his eventual death are well reasoned and well supported. Dr. Wecht's explanation that the miner could have survived the serious cardiac problems longer without the additional burden on the cardiac system of the diseased lungs establishes that pneumoconiosis hastened the miner's death. Dr. Wecht's finding that pneumoconiosis contributed to or hastened the miner's death is also supported by the death certificate statements of Dr. Robinson-Menzie that pneumoconiosis contributed to the miner's death. Therefore, I find Claimant has established the miner's death was due to pneumoconiosis under the provisions of § 718.205(c)(2).

Thus, upon careful consideration of the medical reports of record, I find Claimant has established, by a preponderance of the evidence, that the miner's death was due to pneumoconiosis under the provisions of § 718.205(c)(2). In conclusion, I find the medical reports establish that pneumoconiosis was a substantially contributing cause or factor leading to the miner's death and that pneumoconiosis hastened the miner's death.

## CONCLUSION

Claimant has established that the miner had pneumoconiosis which arose out of his coal mine employment. In addition, Claimant has established the miner's death was due to pneumoconiosis under the provisions of § 718.205(c)(2) because pneumoconiosis was a substantially contributing factor in the miner's death and the evidence established pneumoconiosis hastened the miner's death. Accordingly, Claimant is entitled to survivor's benefits under the Act. Because Claimant's husband, Alphonse R. Haller, died on November 8, 2003, Claimant is entitled to benefits commencing November 1, 2003. 20 C.F.R. § 725.503(c).

## ATTORNEY FEE

No award of attorney's fees for services to Claimant is made herein because no fee application has been received. Thirty (30) days is hereby allowed Claimant's counsel for the submission of a fee application which must conform to §§ 725.365 and 725.366 of the regulations. A service sheet showing that service has been made upon all parties including Claimant must accompany the application. Parties have ten (10) days following receipt of any such application within which to file any objection. The Act prohibits the charging of a fee in the absence of an approved application.

ORDER

It is ordered that the claim of Irma Marie Haller, widow of Alphonse R. Haller, for benefits under the Black Lung Benefits Act is hereby AWARDED. Canterbury Coal Company is ORDERED to pay to Claimant, Irma Marie Haller, all benefits to which she is entitled as the survivor of the deceased miner, Alphonse R. Haller, commencing November 1, 2003.

A

MICHAEL P. LESNIAK
Administrative Law Judge

**NOTICE OF APPEAL RIGHTS:** If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).